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NEWS 1		Web Page for STN Seminar Schedule - N. America
NEWS 2	AUG 10	Time limit for inactive STN sessions doubles to 40 minutes
NEWS 3	AUG 18	COMPENDEX indexing changed for the Corporate Source (CS) field
NEWS 4	AUG 24	ENCOMPLIT/ENCOMPLIT2 reloaded and enhanced
NEWS 5	AUG 24	CA/CAplus enhanced with legal status information for U.S. patents
NEWS 6	SEP 09	50 Millionth Unique Chemical Substance Recorded in CAS REGISTRY
NEWS 7	SEP 11	WPIDS, WPINDEX, and WPIX now include Japanese FTERM thesaurus
NEWS 8	OCT 21	Derwent World Patents Index Coverage of Indian and Taiwanese Content Expanded
NEWS 9	OCT 21	Derwent World Patents Index enhanced with human translated claims for Chinese Applications and Utility Models
NEWS 10	NOV 23	Addition of SCAN format to selected STN databases
NEWS 11	NOV 23	Annual Reload of IFI Databases
NEWS 12	DEC 01	FRFULL Content and Search Enhancements
NEWS 13	DEC 01	DGENE, USGENE, and PCTGEN: new percent identity feature for sorting BLAST answer sets
NEWS 14	DEC 02	Derwent World Patent Index: Japanese FI-TERM thesaurus added
NEWS 15	DEC 02	PCTGEN enhanced with patent family and legal status display data from INPADOCDB
NEWS 16	DEC 02	USGENE: Enhanced coverage of bibliographic and sequence information

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AND CURRENT DISCOVER FILE IS DATED 06 APRIL 2009.

NEWS HOURS STN Operating Hours Plus Help Desk Availability
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FILE 'HOME' ENTERED AT 11:59:36 ON 17 DEC 2009

=> file medline biosis caplus embase
COST IN U.S. DOLLARS                                SINCE FILE      TOTAL
                                                       ENTRY    SESSION
FULL ESTIMATED COST                                     0.22       0.22

FILE 'MEDLINE' ENTERED AT 11:59:57 ON 17 DEC 2009

FILE 'BIOSIS' ENTERED AT 11:59:57 ON 17 DEC 2009
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FILE 'CAPLUS' ENTERED AT 11:59:57 ON 17 DEC 2009
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FILE 'EMBASE' ENTERED AT 11:59:57 ON 17 DEC 2009
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=> s SCHAEBITZ W?/AU
L1          127 SCHAEBITZ W?/AU

=> s l1 and (tbi or traumatic(w)brain(w)injury)
L2            0 L1 AND (TBI OR TRAUMATIC(W) BRAIN(W) INJURY)

=> s l1 and brain
L3           93 L1 AND BRAIN

=> s l3 and stroke
L4           58 L3 AND STROKE

=> dup rem l4
PROCESSING COMPLETED FOR L4
L5           38 DUP REM L4 (20 DUPLICATES REMOVED)

=> s l5 and pd<2002
   1 FILES SEARCHED...
L6            5 L5 AND PD<2002

=> SCHNEIDER A?/AU
SCHNEIDER IS NOT A RECOGNIZED COMMAND
The previous command name entered was not recognized by the system.
For a list of commands available to you in the current file, enter
"HELP COMMANDS" at an arrow prompt (=>).

=> s SCHNEIDER A?/AU
L7         7296 SCHNEIDER A?/AU

=> s l7 and (traumatic(w)brain or tbi)
L8            7 L7 AND (TRAUMATIC(W) BRAIN OR TBI)

=> dup rem l8
PROCESSING COMPLETED FOR L8
L9            3 DUP REM L8 (4 DUPLICATES REMOVED)

=> s KRUEGER C?/AU
L10        1661 KRUEGER C?/AU

=> s l10 and (traumatic(w)brain or tbi)
L11           0 L10 AND (TRAUMATIC(W) BRAIN OR TBI)
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=> s l10 and (brain)
L12         54 L10 AND (BRAIN)

=> s l12 and stroke
L13         17 L12 AND STROKE

=> s l13 and pd<2002
   2 FILES SEARCHED...
L14          0 L13 AND PD<2002

=> dup rem l13
PROCESSING COMPLETED FOR L13
L15          10 DUP REM L13 (7 DUPLICATES REMOVED)

=> s SOMMER C?/AU
L16       1395 SOMMER C?/AU

=> s l16 and (traumatic(w)brain or tbi)
L17          0 L16 AND (TRAUMATIC(W) BRAIN OR TBI)

=> s l16 and (brain or stroke)
L18        271 L16 AND (BRAIN OR STROKE)

=> s l18 and pd<2002
   2 FILES SEARCHED...
L19         74 L18 AND PD<2002

=> dup rem l19
PROCESSING COMPLETED FOR L19
L20          35 DUP REM L19 (39 DUPLICATES REMOVED)

=> s SCHWAB S?/au
L21       1905 SCHWAB S?/AU

=> s l21 and (traumatic(w)brain or tbi)
L22          8 L21 AND (TRAUMATIC(W) BRAIN OR TBI)

=> dup rem l22
PROCESSING COMPLETED FOR L22
L23           5 DUP REM L22 (3 DUPLICATES REMOVED)

=> s KOLLMAR R?/AU
L24        201 KOLLMAR R?/AU

=> s l24 and (traumatic(w)brain or tbi)
L25          3 L24 AND (TRAUMATIC(W) BRAIN OR TBI)

=> dup rem l25
PROCESSING COMPLETED FOR L25
L26           1 DUP REM L25 (2 DUPLICATES REMOVED)

=> s MAURER M?/au
L27       2163 MAURER M?/AU

=> s l27 and (traumatic(w)brain or tbi)
L28          1 L27 AND (TRAUMATIC(W) BRAIN OR TBI)

=> s WEBER D?/au
L29       5346 WEBER D?/AU

=> s l29 and (traumatic(w)brain or tbi)
```

```
L30          6 L29 AND (TRAUMATIC(W) BRAIN OR TBI)

=> dup rem l30
PROCESSING COMPLETED FOR L30
L31           5 DUP REM L30 (1 DUPLICATE REMOVED)

=> s GASSLER N?/au
L32         302 GASSLER N?/AU

=> s l32 and (traumatic(w)brain or tbi)
L33           0 L32 AND (TRAUMATIC(W) BRAIN OR TBI)

=> s l32 and (brain)
L34          43 L32 AND (BRAIN)

=> dup rem l34
PROCESSING COMPLETED FOR L34
L35          21 DUP REM L34 (22 DUPLICATES REMOVED)

=> s l35 and pd<2002
   2 FILES SEARCHED...
L36           0 L35 AND PD<2002

=> dis his

     (FILE 'HOME' ENTERED AT 11:59:36 ON 17 DEC 2009)

     FILE 'MEDLINE, BIOSIS, CAPLUS, EMBASE' ENTERED AT 11:59:57 ON 17 DEC 2009
L1          127 S SCHAEBITZ W?/AU
L2            0 S L1 AND (TBI OR TRAUMATIC(W)BRAIN(W)INJURY)
L3           93 S L1 AND BRAIN
L4           58 S L3 AND STROKE
L5           38 DUP REM L4 (20 DUPLICATES REMOVED)
L6            5 S L5 AND PD<2002
L7         7296 S SCHNEIDER A?/AU
L8            7 S L7 AND (TRAUMATIC(W)BRAIN OR TBI)
L9            3 DUP REM L8 (4 DUPLICATES REMOVED)
L10        1661 S KRUEGER C?/AU
L11           0 S L10 AND (TRAUMATIC(W)BRAIN OR TBI)
L12          54 S L10 AND (BRAIN)
L13          17 S L12 AND STROKE
L14           0 S L13 AND PD<2002
L15          10 DUP REM L13 (7 DUPLICATES REMOVED)
L16        1395 S SOMMER C?/AU
L17           0 S L16 AND (TRAUMATIC(W)BRAIN OR TBI)
L18         271 S L16 AND (BRAIN OR STROKE)
L19          74 S L18 AND PD<2002
L20          35 DUP REM L19 (39 DUPLICATES REMOVED)
L21        1905 S SCHWAB S?/AU
L22           8 S L21 AND (TRAUMATIC(W)BRAIN OR TBI)
L23           5 DUP REM L22 (3 DUPLICATES REMOVED)
L24         201 S KOLLMAR R?/AU
L25           3 S L24 AND (TRAUMATIC(W)BRAIN OR TBI)
L26           1 DUP REM L25 (2 DUPLICATES REMOVED)
L27        2163 S MAURER M?/AU
L28           1 S L27 AND (TRAUMATIC(W)BRAIN OR TBI)
L29        5346 S WEBER D?/AU
L30           6 S L29 AND (TRAUMATIC(W)BRAIN OR TBI)
L31           5 DUP REM L30 (1 DUPLICATE REMOVED)
L32         302 S GASSLER N?/AU
L33           0 S L32 AND (TRAUMATIC(W)BRAIN OR TBI)
L34          43 S L32 AND (BRAIN)
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```
L35          21 DUP REM L34 (22 DUPLICATES REMOVED)
L36           0 S L35 AND PD<2002

=> dis ibib abs 16 1-5

L6   ANSWER 1 OF 5     MEDLINE on STN
ACCESSION NUMBER:   2001069762     MEDLINE
DOCUMENT NUMBER:    PubMed ID: 11088079
TITLE:              An overview of acute stroke therapy: past,
                    present, and future.
AUTHOR:             Fisher M; Schaebitz W
CORPORATE SOURCE:   Department of Neurology, University of Massachusetts
                    Medical School, Worcester, USA.
SOURCE:             Archives of internal medicine, (2000 Nov 27) Vol.
                    160, No. 21, pp. 3196-206.  Ref: 129
                    Journal code: 0372440. ISSN: 0003-9926.
PUB. COUNTRY:       United States
DOCUMENT TYPE:      Journal; Article; (JOURNAL ARTICLE)
                    General Review; (REVIEW)
LANGUAGE:           English
FILE SEGMENT:       Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH:        200101
ENTRY DATE:         Entered STN: 22 Mar 2001
                    Last Updated on STN: 22 Mar 2001
                    Entered Medline: 4 Jan 2001

L6   ANSWER 2 OF 5  BIOSIS  COPYRIGHT (c) 2009 The Thomson Corporation  on STN
ACCESSION NUMBER:   2002:79559  BIOSIS
DOCUMENT NUMBER:    PREV200200079559
TITLE:              The role of stroke MRI in intracranial and
                    subarachnoid hemorrhage.
                    Original Title: Stellenwert des Schlaganfall-MRT bei
                    intrazerebralen und subarachnoidalen Blutungen.
AUTHOR(S):          Schellinger, P. D. [Reprint author]; Fiebach, J.; Mohr, A.;
                    Kollmar, R.; Schwarz, S.; Schaebitz, W. R.;
                    Sartor, K.; Hacke, W.
CORPORATE SOURCE:   Neurologische Klinik, Kopfklinik, Universitaetsklinikums
                    Heidelberg, Im Neuenheimer Feld 400, 69120, Heidelberg,
                    Germany
                    Peter_Schellinger@med.uni-heidelberg.de
SOURCE:             Nervenarzt, (Dezember, 2001) Vol. 72, No. 12, pp.
                    907-917. print.
                    CODEN: NERVAF. ISSN: 0028-2804.
DOCUMENT TYPE:      Article
                    General Review; (Literature Review)
LANGUAGE:           German
ENTRY DATE:         Entered STN: 16 Jan 2002
                    Last Updated on STN: 25 Feb 2002
AB   Intracranial hemorrhage (ICH) accounts for 15% of all strokes.
     In hyperacute emergency assessment, CT is the diagnostic standard for
     differentiating between hyperacute ICH and ischemic stroke.  At
     this stage, MRI is considered to be of little value for the diagnosis of
     ICH or subarachnoidal hemorrhage (SAH).  We review the current literature
     and characterize the role of MRI in the diagnosis of ICH and SAH as well
     as hyperacute stroke in general: While MRI is considered
     superior to CT in the diagnosis of subacute and chronic ICH/SAH, in
     hyperacute ICH this is still a matter of debate.  MRI signal
     characteristics of ICH depend on hemoglobin degradation.  Deoxyhemoglobin
     is the MRI substrate for demonstration of blood due to its paramagnetic
     properties causing signal loss on susceptibility weighted images (T2*-WI).
     Preliminary data, however, suggest that the sensitivity of modern
     stroke MRI protocols is sufficiently high for hyperacute ICH and
```

SAH and may render additional information with regard to the etiology of ICH or SAH. Further interest is focused on perihemorrhagic pathophysiologic processes, which may help to improve therapeutic decision making in patients with ICH.

L6 ANSWER 3 OF 5 BIOSIS COPYRIGHT (c) 2009 The Thomson Corporation on STN
ACCESSION NUMBER: 2001:574299 BIOSIS
DOCUMENT NUMBER: PREV200100574299
TITLE: Recombinant granulocyte-colony stimulating factor (rG-CSF) is neuroprotective following focal transient cerebral ischemia and excitotoxity.
AUTHOR(S): Schaebitz, W. R. [Reprint author]; Kollmar, R. [Reprint author]; Schwaninger, M. [Reprint author]; Sommer, C.; Schoelzke, M. [Reprint author]; Schwab, S. [Reprint author]; Wildemann, B. [Reprint author]
CORPORATE SOURCE: Neurology, University of Heidelberg, Heidelberg, Germany
SOURCE: Society for Neuroscience Abstracts, (2001) Vol. 27, No. 2, pp. 2027. print.
Meeting Info.: 31st Annual Meeting of the Society for Neuroscience. San Diego, California, USA. November 10-15, 2001.
ISSN: 0190-5295.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 12 Dec 2001
Last Updated on STN: 25 Feb 2002

AB Granulocyte-Colony Stimulating Factor (G-CSF) may influence the pathogenesis of cerebral ischemia by several mechanisms. Therefore, the effects of recombinant G-CSF (rG-CSF) were investigated in transient focal cerebral ischemia and a model of excitotoxicity. Methods: Male wistar rats underwent transient middle cerebral artery occlusion (MCAO) for 90 min. 30 min after induction of ischemia, the treatment group (n=12) received 60 microg/kg body weight of rG-CSF intravenously for 90 min (Control: n=12, MCAO 90 min, saline i.v.). At 24 hours the cerebral infarct and edema volume were calculated from TTC-stained brain slices. The effects of rG-CSF on glutamate-induced neuronal death were studied in cell culture, and the expression of G-CSF-receptors in the brain was investigated by RT-PCR, immunohistochemistry and immunoblotting. Results: RG-CSF reduced the infarct volume to 100,95 mm3+-36 mm3 vs 194,28 mm3+-66,65 mm3 ($p<0.01$) in the control and the cerebral edema by 60,02% ($p<0.05$). By immunostaining, G-CSF receptors were detectable in neurons and glial cells. The expression of a G-CSF-receptor in the brain was verified by Westernblotting and RT-PCR. In cell culture, rG-CSF protected primarily neurons. As shown here, rG-CSF has neuroprotective properties after stroke, that are probably receptor mediated, and reduced glutamate induced toxicity.

L6 ANSWER 4 OF 5 BIOSIS COPYRIGHT (c) 2009 The Thomson Corporation on STN
ACCESSION NUMBER: 2001:337477 BIOSIS
DOCUMENT NUMBER: PREV200100337477
TITLE: Hemorrhagic transformation of ischemic brain tissue: Asymptomatic or symptomatic?.
AUTHOR(S): Berger, Christian [Reprint author]; Fiorelli, Marco; Steiner, Thorsten; Schaebitz, Wolf-Ruediger; Bozzao, Luigi; Bluhmki, Erich; Hacke, Werner; von Kummer, Ruediger
CORPORATE SOURCE: Department of Neurology, University of Heidelberg, Im Neuenheimer Feld 400, D-69221, Heidelberg, Germany
christian_berger@med.uni-heidelberg.de
SOURCE: Stroke, (June, 2001) Vol. 32, No. 6, pp. 1330-1335. print.

CODEN: SJCCA7. ISSN: 0039-2499.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 18 Jul 2001
Last Updated on STN: 19 Feb 2002

AB Background and Purpose: The term symptomatic hemorrhage secondary to ischemic stroke implies a clear causal relationship between clinical deterioration and hemorrhagic transformation (HT) regardless of the type of HT. The aim of this study was to assess which type of HT independently affects clinical outcome. Methods: We used the data set of the European Cooperative Acute Stroke Study (ECASS) II for a post hoc analysis. All patients had a control CT scan after 24 to 96 hours or earlier in case of rapid and severe clinical deterioration. HT was categorized according to radiological criteria: hemorrhagic infarction type 1 and type 2 and parenchymal hematoma type 1 and type 2. The clinical course was prospectively documented with the National Institutes of Health Stroke Scale and the modified Rankin Scale. The independent risk of each type of HT was calculated for clinical deterioration at 24 hours and disability and death at 3 months after stroke onset and adjusted for possible confounding factors such as age, severity of stroke syndrome at baseline, and extent of the ischemic lesion on the initial CT. Results: Compared with absence of HT, only parenchymal hematoma type 2 was associated with an increased risk for deterioration at 24 hours after stroke onset (adjusted odds ratio, 18; 95% CI, 6 to 56) and for death at 3 months (adjusted odds ratio, 11; 95% CI, 3.7 to 36). All other types of HT did not independently increase the risk of late deterioration. Conclusions: Only parenchymal hematoma type 2 independently causes clinical deterioration and impairs prognosis. It has a distinct radiological feature: it is a dense homogeneous hematoma >30% of the ischemic lesion volume with significant space-occupying effect.

L6 ANSWER 5 OF 5 BIOSIS COPYRIGHT (c) 2009 The Thomson Corporation on STN
ACCESSION NUMBER: 2000:310578 BIOSIS
DOCUMENT NUMBER: PREV200000310578
TITLE: Effect of propentofylline (HWA 285) on focal ischemia in rats: Effect of treatment and posttreatment duration on infarct size.
AUTHOR(S): Haag, Peter [Reprint author]; Schneider, Tom; Schaebitz, Wolf-Ruediger; Hacke, Werner
CORPORATE SOURCE: Department of Neurology, Klinikum Ingolstadt, Krumenauerstrasse 25, 85049, Ingolstadt, Germany
SOURCE: Journal of the Neurological Sciences, (April 1, 2000) Vol. 175, No. 1, pp. 52-56. print.
CODEN: JNSCAG. ISSN: 0022-510X.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 19 Jul 2000
Last Updated on STN: 7 Jan 2002

AB Background and purpose: in this study we tested the potentially neuroprotective properties of propentofylline in a model of focal ischemia with long-term, repeated treatment. Methods: 37 male Wistar rats (280-300 g) underwent permanent occlusion of the middle cerebral artery (MCA). Infusion was started 30 min after occlusion of the MCA over a period of 2 h with a dosage of 0.01 mg/kg body weight. Immediately after the termination of infusion repetitive intraperitoneal injections were started. Animals were assigned to four groups: continuous treatment for a period of 12 h with 24-h survival (group A, n = 9) or 48-h survival (group B, n = 10), continuous treatment for a period of 48 h with 48-h survival (group C, n=9) and placebo (group D, n=9). Infarct size was calculated from brain slices stained with 2,3,5-triphenyltetrazolium chloride. Results: the infarct size was significantly reduced in group C

(treatment for 48 h) (163.9 +- 30.5 mm3) compared to the placebo group (297.4 +- 17.7 mm3). No effect on infarct size was observed in group A (196.8 +- 37.3 mm3) and group B (239.6 +- 42.9 mm3) compared to placebo. Conclusion: continuous i.p. injections of propentofylline over a period of 48 h significantly reduces infarct size in an animal model of focal cerebral ischemia. With shorter periods of continuous administration of the drug and delayed postmortem analysis, reductions in the infarct size did not reach a level of significance. These data show the importance of continuous long-term administration after ischemic stroke in clinical trials to achieve the beneficial effects of neuroprotection by propentofylline.

=> dis ibib abs l9 1-3

L9 ANSWER 1 OF 3 MEDLINE on STN DUPLICATE 1
ACCESSION NUMBER: 2008127990 MEDLINE
DOCUMENT NUMBER: PubMed ID: 18246320
TITLE: [Therapeutic hypothermia].
Therapeutische Hypothermie.
AUTHOR: Schneider A; Popp E; Teschendorf P; Bottiger B W
CORPORATE SOURCE: Klinik fur Anasthesiologie und Operative Intensivmedizin, Klinikum der Universitat zu Koln, Koln..
andreas.schneider@uk-koeln.de
SOURCE: Der Anaesthesist, (2008 Feb) Vol. 57, No. 2, pp. 197-206; quiz 207-8. Ref: 91
Journal code: 0370525. ISSN: 0003-2417.
PUB. COUNTRY: Germany: Germany, Federal Republic of
DOCUMENT TYPE: (ENGLISH ABSTRACT)
Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
LANGUAGE: German
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200810
ENTRY DATE: Entered STN: 22 Feb 2008
Last Updated on STN: 3 Nov 2008
Entered Medline: 31 Oct 2008
AB The use of therapeutic hypothermia has been shown to improve survival and neurological outcome following cardiac arrest. Patients with traumatic brain injury or ischemic stroke also responded positively to therapeutic hypothermia, which may be induced by various procedures including surface cooling, endovascular cooling catheter and cold infusion. Possible side effects include infection and hemorrhage, as well as changes in water and electrolyte levels. It is the aim of this article to provide an overview of studies to date, as well as practical guidance for the application of therapeutic hypothermia.

L9 ANSWER 2 OF 3 BIOSIS COPYRIGHT (c) 2009 The Thomson Corporation on STN
ACCESSION NUMBER: 2007:306868 BIOSIS
DOCUMENT NUMBER: PREV200700296277
TITLE: Embryonic stem cell transplantation after experimental traumatic brain injury dramatically
improves neurological outcome, but may cause tumors (vol 24, pg 216, 2007).
AUTHOR(S): Riess, P.; Molcanyi, M.; Bentz, K.; Maegele, M.; Simanski, C.; Carlitscheck, C.; Schneider, A.; Hescheler, J.; Bouillon, B.; Schaefer, U.; Neugebauer, E.
SOURCE: Journal of Neurotrauma, (FEB 2007) Vol. 24, No. 2, pp. 433. ISSN: 0897-7151.
DOCUMENT TYPE: Article
Errata
LANGUAGE: English

ENTRY DATE: Entered STN: 9 May 2007
Last Updated on STN: 9 May 2007

L9 ANSWER 3 OF 3 MEDLINE on STN DUPLICATE 2
ACCESSION NUMBER: 2007059406 MEDLINE
DOCUMENT NUMBER: PubMed ID: 17263685
TITLE: Embryonic stem cell transplantation after experimental traumatic brain injury dramatically improves neurological outcome, but may cause tumors.
AUTHOR: Riess Peter; Molcanyi Marek; Bentz Kristine; Maegele Mark; Simanski Christian; Carlitscheck Christoph; Schneider Annette; Hescheler Jurgen; Bouillon Bertil; Schafer Ute; Neugebauer Edmund
CORPORATE SOURCE: Department of Trauma and Orthopedic Surgery, University of Witten/Herdecke, Cologne Merheim Medical Center, Ostmerheimerstrasse 200, 51109 Cologne, Germany.
SOURCE: Journal of neurotrauma, (2007 Jan) Vol. 24, No. 1, pp. 216-25.
Journal code: 8811626. ISSN: 0897-7151.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200703
ENTRY DATE: Entered STN: 1 Feb 2007
Last Updated on STN: 15 Mar 2007
Entered Medline: 14 Mar 2007

AB Transplantation of embryonic stem (ES) cells may provide cures for the damaged nervous system. Pre-differentiated ES or neuronal precursor cells have been investigated in various animal models of neurodegenerative diseases including traumatic brain injury (TBI). To our knowledge, no study has yet examined the effects of undifferentiated, murine ES cells on functional recovery and tumorigenity following implantation into injured rat brains. We evaluated the effect of transplantation of undifferentiated, murine embryonic cells on the recovery of motor function following lateral fluid percussion brain injury in Sprague-Dawley rats. At 3 days post-injury, animals received stereotactic injections of either embryonic stem cell suspension or injections of phosphate buffered saline without cells (control) into the injured cortex. Neurological motor function assessments were performed before injury, 72 h, 1, 3, and 6 weeks after transplantation using a Rotatrod and a Composite Neuroscore test. During this time period brain injured animals receiving ES cell transplantation showed a significant improvement in the Rotarod Test and in the Composite Neuroscore Test as compared to phosphate buffered saline (PBS)-treated animals. At 1 week post-transplantation, ES cells were detectable in 100% of transplanted animals. At 7 weeks following transplantation, EScells were detectable in only one animal. Two of 10 xenotransplanted animals revealed tumor formation over the observation period. These findings provide evidence for therapeutic potency of embryonic stem cell transplantation after TBI in rat, but also raise serious safety concerns about the use of such cells in human.

=> dis ibib abs l23 1-5

L23 ANSWER 1 OF 5 BIOSIS COPYRIGHT (c) 2009 The Thomson Corporation on STN
ACCESSION NUMBER: 2009:617954 BIOSIS
DOCUMENT NUMBER: PREV200900619057
TITLE: Patients with mild traumatic brain injury have subtle autonomic cardiovascular dysfunction with ocular pressure test.

AUTHOR(S): Hilz, M. J. [Reprint Author]; Aurnhammer, F.; Anders, S.; Marthol, H.; Blaszczynska, P.; Schroeder, T.; Rossmeissl, A.; Schwab, S.; Flanagan, S.; De Fina, P.
CORPORATE SOURCE: Univ Erlangen Nurnberg, Dept Neurol, D-8520 Erlangen, Germany
SOURCE: European Journal of Neurology, (OCT 2009) Vol. 16, No. Suppl. 3, pp. 375.
Meeting Info.: 13th Congress of the European-Federation-of-Neurological-Societies. Florence, ITALY. September 12 -15, 2009. European Federat Neurol Soc.
ISSN: 1351-5101.
DOCUMENT TYPE: Conference; (Meeting)
Conference; (Meeting Poster)
LANGUAGE: English
ENTRY DATE: Entered STN: 12 Nov 2009
Last Updated on STN: 12 Nov 2009

L23 ANSWER 2 OF 5 BIOSIS COPYRIGHT (c) 2009 The Thomson Corporation on STN
ACCESSION NUMBER: 2009:617295 BIOSIS
DOCUMENT NUMBER: PREV200900618398
TITLE: Patients with mild traumatic brain injury show subtle sympathetic cardiac dysfunction during orthostatic challenge.
AUTHOR(S): Hilz, M. J. [Reprint Author]; Anders, S.; Aurnhammer, F.; Marthol, H.; Baltadzhieva, R.; Schroeder, T.; Rossmeissl, A.; Schwab, S.; Flanagan, S.; De Fina, P.
CORPORATE SOURCE: Univ Erlangen Nurnberg, Dept Neurol, Erlangen, Germany
SOURCE: European Journal of Neurology, (OCT 2009) Vol. 16, No. Suppl. 3, pp. 53.
Meeting Info.: 13th Congress of the European-Federation-of-Neurological-Societies. Florence, ITALY. September 12 -15, 2009. European Federat Neurol Soc.
ISSN: 1351-5101.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 12 Nov 2009
Last Updated on STN: 12 Nov 2009

L23 ANSWER 3 OF 5 MEDLINE on STN DUPLICATE 1
ACCESSION NUMBER: 2007741382 MEDLINE
DOCUMENT NUMBER: PubMed ID: 17828037
TITLE: Differential recovery of behavioral status and brain function assessed with functional magnetic resonance imaging after mild traumatic brain injury in the rat.
AUTHOR: Henninger Nils; Sicard Kenneth M; Li Zhixin; Kulkarni Praveen; Dutzmann Stephan; Urbanek Christian; Schwab Stefan; Fisher Marc
CORPORATE SOURCE: Department of Internal Medicine, University of Massachusetts Medical School, Worcester, MA, USA.. henningn@ummhc.org
SOURCE: Critical care medicine, (2007 Nov) Vol. 35, No. 11, pp. 2607-14.
Journal code: 0355501. ISSN: 0090-3493.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 200802
ENTRY DATE: Entered STN: 18 Dec 2007
Last Updated on STN: 16 Feb 2008

Entered Medline: 15 Feb 2008

AB OBJECTIVE: The relationship between cerebral integrity, recovery of brain function, and neurologic status after mild traumatic brain injury is incompletely characterized. DESIGN: Prospective and randomized study in rodents. SETTING: University laboratory. SUBJECTS: Male Wistar rats (290-310 g). INTERVENTIONS: In rats, quantitative diffusion weighted imaging (DWI), perfusion weighted imaging (PWI), T2-weighted imaging (T2WI), and functional magnetic resonance imaging (fMRI) were performed up to 21 days after weight-induced, closed-head, mild traumatic brain injury (MTBI, n = 6) or sham operation (n = 6). Pixel-by-pixel analysis and region of interest analysis were used to evaluate structural (apparent diffusion coefficient [ADC] and basal cerebral blood flow [bCBF]) and functional magnetic resonance signal changes within the brain, respectively. Quantitative fMRI signal changes were correlated with behavioral measures. MEASUREMENTS AND MAIN RESULTS: Despite normal appearing DWI and T2WI findings following MTBI, persistent hypoperfusion developed that was not associated with cytotoxic edema. In contrast, the ADC was significantly increased by approximately 5% at 1 and 7 days post-MTBI. Post-MTBI fMRI responses to hypercapnia and forepaw stimulation were significantly impaired and showed a differential recovery rate between and within investigated region of interests. Significant dysfunction in forepaw placement test persisted up to day 1 and correlated significantly with fMRI signal changes in the primary somatosensory and motor cortices. CONCLUSIONS: MTBI produced distinct changes on multimodal MRI and behavioral variables acutely and chronically. Following MTBI, fMRI and ADC-bCBF pixel-by-pixel analysis identified subtle structural and functional alterations in the brain that appeared completely normal on conventional DWI and T2WI after concussion injury. The former techniques may therefore provide great potential for understanding mild traumatic brain injury, identifying mechanisms underlying recovery, and investigating specific interventions to enhance functional outcome.

L23 ANSWER 4 OF 5 MEDLINE on STN DUPLICATE 2
ACCESSION NUMBER: 2005494171 MEDLINE
DOCUMENT NUMBER: PubMed ID: 16084512
TITLE: Impaired spatial learning in a novel rat model of mild cerebral concussion injury.
AUTHOR: Henninger Nils; Dutzmann Stephan; Sicard Kenneth M; Kollmar Rainer; Bardutzky Jurgen; Schwab Stefan
CORPORATE SOURCE: Department of Neurology, University of Heidelberg, 69120 Heidelberg, Germany.. nils.henninger@umassmed.edu
SOURCE: Experimental neurology, (2005 Oct) Vol. 195, No. 2, pp. 447-57.
Journal code: 0370712. ISSN: 0014-4886.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200511
ENTRY DATE: Entered STN: 17 Sep 2005
Last Updated on STN: 15 Dec 2005
Entered Medline: 21 Nov 2005

AB The aim of the present study was to develop a model of mild traumatic brain injury in the rat that mimics human concussive brain injury suitable to study pathophysiology and potential treatments. 34 male Wistar rats received a closed head trauma (TBI) and 30 animals served as controls (CON). Immediately following trauma, animals lost their muscle tone and righting reflex response, recovering from the latter within 11.4 +/- 8.2 min. Corneal reflex and whisker responses returned within 4.5 +/- 3.0 min and 6.1 +/- 2.9 min,

respectively. The impact resulted in a short transient decrease of pO2 (P < 0.001), increase in mean arterial blood pressure (P = 0.026), and a reduction of heart rate (P < 0.01). Serial MRI did not show any abnormalities across the entire cerebrum on diffusion, T1, T2, and T2*-weighted images at all investigated time points. TBI animals needed significantly longer to locate the hidden platform in a Morris water maze and spent less time in the training quadrant than controls. TBI led to a significant neuronal loss in frontal cortex (P < 0.001), as well as hippocampal CA3 (P = 0.017) and CA1 (P = 0.002) at 9 days after the trauma; however, cytoskeletal architecture was preserved as indicated by normal betaAPP- and MAP-2 staining. We present a unique, noninvasive rat model of mild closed head trauma with characteristics of human concussion injury, including brief loss of consciousness, cognitive impairment, and minor brain injury.

L23 ANSWER 5 OF 5 BIOSIS COPYRIGHT (c) 2009 The Thomson Corporation on STN
ACCESSION NUMBER: 2002:292724 BIOSIS
DOCUMENT NUMBER: PREV200200292724
TITLE: Use of hypertonic saline in ischemic stroke: Response.
AUTHOR(S): Schwarz, Stefan [Reprint author]; Georgiadis, Dimitrios [Reprint author]; Schwab, Stefan [Reprint author]; Aschoff, Alfred
CORPORATE SOURCE: Department of Neurology, University of Heidelberg, Heidelberg, Germany
SOURCE: Stroke, (April, 2002) Vol. 33, No. 4, pp. 1167. print.
CODEN: SJCCA7. ISSN: 0039-2499.
DOCUMENT TYPE: Letter
LANGUAGE: English
ENTRY DATE: Entered STN: 15 May 2002
Last Updated on STN: 15 May 2002

=> dis ibib abs l26

L26 ANSWER 1 OF 1 MEDLINE on STN DUPLICATE 1
ACCESSION NUMBER: 2005494171 MEDLINE
DOCUMENT NUMBER: PubMed ID: 16084512
TITLE: Impaired spatial learning in a novel rat model of mild cerebral concussion injury.
AUTHOR: Henninger Nils; Dutzmann Stephan; Sicard Kenneth M; Kollmar Rainer; Bardutzky Jurgen; Schwab Stefan
CORPORATE SOURCE: Department of Neurology, University of Heidelberg, 69120 Heidelberg, Germany.. nils.henninger@umassmed.edu
SOURCE: Experimental neurology, (2005 Oct) Vol. 195, No. 2, pp. 447-57.
Journal code: 0370712. ISSN: 0014-4886.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200511
ENTRY DATE: Entered STN: 17 Sep 2005
Last Updated on STN: 15 Dec 2005
Entered Medline: 21 Nov 2005
AB The aim of the present study was to develop a model of mild traumatic brain injury in the rat that mimics human concussive brain injury suitable to study pathophysiology and potential treatments. 34 male Wistar rats received a closed head trauma (TBI) and 30 animals served as controls (CON). Immediately following trauma, animals lost their muscle tone and righting reflex response, recovering from the latter within 11.4 +/- 8.2 min. Corneal reflex and whisker responses returned within 4.5 +/- 3.0 min and 6.1 +/- 2.9 min,

respectively. The impact resulted in a short transient decrease of pO2 ($P < 0.001$), increase in mean arterial blood pressure ($P = 0.026$), and a reduction of heart rate ($P < 0.01$). Serial MRI did not show any abnormalities across the entire cerebrum on diffusion, T1, T2, and T2*-weighted images at all investigated time points. TBI animals needed significantly longer to locate the hidden platform in a Morris water maze and spent less time in the training quadrant than controls. TBI led to a significant neuronal loss in frontal cortex ($P < 0.001$), as well as hippocampal CA3 ($P = 0.017$) and CA1 ($P = 0.002$) at 9 days after the trauma; however, cytoskeletal architecture was preserved as indicated by normal betaAPP- and MAP-2 staining. We present a unique, noninvasive rat model of mild closed head trauma with characteristics of human concussion injury, including brief loss of consciousness, cognitive impairment, and minor brain injury.

=> dis ibib abs l28

L28 ANSWER 1 OF 1 EMBASE COPYRIGHT (c) 2009 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 2008144852 EMBASE
TITLE: Proteomics of human cerebral microdialysate: From detection of biomarkers to clinical application.
AUTHOR: Maurer, Martin H., Dr. (correspondence)
CORPORATE SOURCE: Department of Physiology and Pathophysiology, University of Heidelberg, Heidelberg, Germany. maurer@sygnis.de
AUTHOR: Haux, Daniel; Unterberg, Andreas W.; Sakowitz, Oliver W.
CORPORATE SOURCE: Department of Neurosurgery, University of Heidelberg, Heidelberg, Germany.
AUTHOR: Maurer, Martin H., Dr. (correspondence)
CORPORATE SOURCE: SYGNIS Bioscience, Im Neuenheimer Feld 515, 69120 Heidelberg, Germany. maurer@sygnis.de
SOURCE: Proteomics - Clinical Applications, (Mar 2008) Vol. 2, No. 3, pp. 437-443.
Refs: 43
ISSN: 1862-8346
COUNTRY: Germany
DOCUMENT TYPE: Journal; General Review; (Review)
FILE SEGMENT: 008 Neurology and Neurosurgery
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 4 Apr 2008
Last Updated on STN: 4 Apr 2008

AB Cerebral microdialysis is applied in clinical neurology and neurosurgery as monitoring tool in patients to evaluate the progression of severe diseases, such as stroke or trauma. Besides small molecules, e.g. metabolites and neurotransmitters, also the macromolecules, such as proteins and larger chemical compounds cross the dialysis membrane of the catheters implanted into the human brain parenchyma. Microdialysis can be used to extract molecules from the extracellular space of the brain in vivo, but additionally to deliver drugs, since the exchange is dependent on concentration gradients. Cerebral microdialysis may also be useful in the prediction of the clinical onset of symptoms, based on changes in the composition of pre-symptomatic microdialysate. For example, symptomatic vasospasm, which is a complication after subarachnoid hemorrhage, may be predicted by the combination of cerebral microdialysis and a proteomics approach. We will introduce the basic concepts of cerebral microdialysis, discuss possible clinical applications, and evaluate the application of proteomic approaches. With regard to technological aspects, we describe two-dimensional gel electrophoresis, high-pressure liquid chromatography, and mass spectrometry. With regard to clinical aspects, we discuss ethics, feasibility, time-course, and therapeutic options. In conclusion,

proteomics of cerebral microdialysate may be used for diagnosis, disease monitoring, and therapeutic intervention of neurological patients. .COPYRGT. 2008 Wiley-VCH Verlag GmbH & Co. KGaA, Weinheim.

=> dis ibib abs l31

L31 ANSWER 1 OF 5 BIOSIS COPYRIGHT (c) 2009 The Thomson Corporation on STN
ACCESSION NUMBER: 2009:259989 BIOSIS
DOCUMENT NUMBER: PREV200900259989
TITLE: Reduced-Intensity Regimens for Allogeneic Stem Cell Transplantation Improve the Outcome in Advanced Multiple Myeloma.
AUTHOR(S): Cole, Suzanne M. [Reprint Author]; Saliba, Rima; Pelosini, Matteo; Mendoza, Floralyn L.; Weber, Donna; Wang, Michael; Thomas, Sheeba; Orlowski, Robert Z.; Shah, Jatin; Alousi, Amin; Hosing, Chitra; Popat, Uday; Kebriaei, Partow; Anderlini, Paolo; Khouri, Issa F.; Champlin, Richard; Giralt, Sergio; Qazilbash, Muzaffar H.
CORPORATE SOURCE: Univ Texas MD Anderson Canc Ctr, Houston, TX 77030 USA
SOURCE: Blood, (NOV 16 2008) Vol. 112, No. 11, pp. 1132-1133. Meeting Info.: 50th Annual Meeting of the American-Society-of-Hematology. San Francisco, CA, USA. December 06 -09, 2008. Amer Soc Hematol.
CODEN: BLOOAW. ISSN: 0006-4971.
DOCUMENT TYPE: Conference; (Meeting)
Conference; (Meeting Poster)
LANGUAGE: English
ENTRY DATE: Entered STN: 16 Apr 2009
Last Updated on STN: 16 Apr 2009

AB Background: Allogeneic hematopoietic stem cell transplantation (allo SCT) has two potential advantages over autologous SCT: a tumor-free graft and graft-versus-myeloma (GVM) effect. Alto SCT's potential to induce long term remission has, however, been offset by high rates of transplant-related non-relapse mortality (TRM). Reduced-intensity conditioning (RIC) regimens for allo SCT are associated with lower TRM without compromising the GVM effect.Methods: We, retrospectively analyzed our experience in 69 patients (30 females and 39 males) with heavily pretreated, relapsed myeloma, who received allo SCT at our institution between 1985 and 2007. Eighteen patients received myeloablative regimens (MA), while 51 received RIC regimens. MA regimens were TBI -based in 5 patients, high-dose busulfan-containing in 6 patients and high-dose melphalan containing (180-200 mg/ m(2)) in 7 patients. RIC regimens were a combination of fludarabine (90-120 mg/m(2)) and melphalan (100-140 mg/m(2)). Median age of patients at allo SCT in both groups was 51 years. Median interval from diagnosis to allo SCT was 35.4 months in MA group, and 34.2 months in RIC group. Eight (44%) patients in MA group and 36 (70%) patients in RIC group had prior autologous SCT. Six patients (33%) in the MA group and 11 (25%) in the RIC group received allo SCT from unrelated donors (p=0.3). Median number of prior treatment regimens were 5 (range 1-10) in both groups. Stem cell source was peripheral blood in 3 patients in MA group and 41 patients in the RIC group (p=0.0001).Results: Median follow-up in surviving patients was 27 months (3-98). All patients achieved engraftment. Cumulative TRM at 1 year was 56% in the MA group and 25% in the RIC group (p=0.03). Overall response rates in evaluable patients were 69% (CR=15%, PR= 54%) in MA group, and 79% (CR=23%, PR=56%) in the RIC group (p=0.47). Disease progression at 2 years was seen in 8 patients (44%) in the MA group and 25 patients (49%) in the RIC group (p=0.78). Median progression-free survival (PFS) in MA vs. RIC groups was 4.1 and 6.8 months, respectively (p=0.003) and median overall survival (OS)) in MA vs. RIC group was 5.3 and 13.9 months, respectively (p=0.001). Cumulative Incidence of grade II-IV acute graft-vs.-host

(GVHD) in MA vs. RIC groups disease was 33 vs. 27% (p=0.76); cumulative incidence of chronic GVHD in MA vs. RIC group was 54% vs. 47% (p=0.41) in evaluable patients. At the time of this analysis, 13 patients (25%) were still alive in RIC group, 7 of whom (14%) were in remission for up to 6 years post allo SCT. The most common causes of death were recurrent disease (30 patients; 43%), acute or chronic GVHD (16 patients; 23%) and opportunistic infections (5 patients: 7%).Conclusions: Allo SCT after RIC regimens is associated with longer PFS and OS and lower TRM. There was no increase in the risk of relapse, or acute or chronic GVHD. These regimens can safely replace MA regimens and may offer greater benefit if utilized earlier in the course of disease.[GRAPHICS]

=> logoff
ALL L# QUERIES AND ANSWER SETS ARE DELETED AT LOGOFF
LOGOFF? (Y)/N/HOLD:y

(FILE 'HOME' ENTERED AT 11:59:36 ON 17 DEC 2009)

FILE 'MEDLINE, BIOSIS, CAPLUS, EMBASE' ENTERED AT 11:59:57 ON 17 DEC 2009

L1	127	SEA FILE=MFE SPE=ON ABB=ON PLU=ON	SCHAEBITZ W?/AU
L2	0	SEA FILE=MFE SPE=ON ABB=ON PLU=ON	L1 AND (TBI OR TRAUMATIC(W) BRAIN(W) INJURY)
L3	93	SEA FILE=MFE SPE=ON ABB=ON PLU=ON	L1 AND BRAIN
L4	58	SEA FILE=MFE SPE=ON ABB=ON PLU=ON	L3 AND STROKE
L5	38	DUP REM L4 (20 DUPLICATES REMOVED)	
L*** DEL	1	S L3 AND STROKE	
L*** DEL	29	S L3 AND STROKE	
L*** DEL	27	S L3 AND STROKE	
L*** DEL	27	S L3 AND STROKE	
L6	5	SEA FILE=MFE SPE=ON ABB=ON PLU=ON	L5 AND PD<2002
L7	7296	SEA FILE=MFE SPE=ON ABB=ON PLU=ON	SCHNEIDER A?/AU
L8	7	SEA FILE=MFE SPE=ON ABB=ON PLU=ON	L7 AND (TRAUMATIC(W) BRAIN OR TBI)
L9	3	DUP REM L8 (4 DUPLICATES REMOVED)	
L10	1661	SEA FILE=MFE SPE=ON ABB=ON PLU=ON	KRUEGER C?/AU
L11	0	SEA FILE=MFE SPE=ON ABB=ON PLU=ON	L10 AND (TRAUMATIC(W) BRAIN OR TBI)
L12	54	SEA FILE=MFE SPE=ON ABB=ON PLU=ON	L10 AND (BRAIN)
L13	17	SEA FILE=MFE SPE=ON ABB=ON PLU=ON	L12 AND STROKE
L14	0	SEA FILE=MFE SPE=ON ABB=ON PLU=ON	L13 AND PD<2002
L15	10	DUP REM L13 (7 DUPLICATES REMOVED)	
L16	1395	SEA FILE=MFE SPE=ON ABB=ON PLU=ON	SOMMER C?/AU
L17	0	SEA FILE=MFE SPE=ON ABB=ON PLU=ON	L16 AND (TRAUMATIC(W) BRAIN OR TBI)
L18	271	SEA FILE=MFE SPE=ON ABB=ON PLU=ON	L16 AND (BRAIN OR STROKE)
L19	74	SEA FILE=MFE SPE=ON ABB=ON PLU=ON	L18 AND PD<2002
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L21	1905	SEA FILE=MFE SPE=ON ABB=ON PLU=ON	SCHWAB S?/AU
L22	8	SEA FILE=MFE SPE=ON ABB=ON PLU=ON	L21 AND (TRAUMATIC(W) BRAIN OR TBI)
L23	5	DUP REM L22 (3 DUPLICATES REMOVED)	
L24	201	SEA FILE=MFE SPE=ON ABB=ON PLU=ON	KOLLMAR R?/AU
L25	3	SEA FILE=MFE SPE=ON ABB=ON PLU=ON	L24 AND (TRAUMATIC(W) BRAIN OR TBI)
L26	1	DUP REM L25 (2 DUPLICATES REMOVED)	
L27	2163	SEA FILE=MFE SPE=ON ABB=ON PLU=ON	MAURER M?/AU
L28	1	SEA FILE=MFE SPE=ON ABB=ON PLU=ON	L27 AND (TRAUMATIC(W) BRAIN OR TBI)
L29	5346	SEA FILE=MFE SPE=ON ABB=ON PLU=ON	WEBER D?/AU
L30	6	SEA FILE=MFE SPE=ON ABB=ON PLU=ON	L29 AND (TRAUMATIC(W)

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                BRAIN OR TBI)
L31             5 DUP REM L30 (1 DUPLICATE REMOVED)
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L33             0 SEA FILE=MFE SPE=ON  ABB=ON  PLU=ON  L32 AND (TRAUMATIC(W)
                BRAIN OR TBI)
L34            43 SEA FILE=MFE SPE=ON  ABB=ON  PLU=ON  L32 AND (BRAIN)
L35            21 DUP REM L34 (22 DUPLICATES REMOVED)
L*** DEL        9 S L32 AND (BRAIN)
L*** DEL       12 S L32 AND (BRAIN)
L*** DEL       13 S L32 AND (BRAIN)
L*** DEL        9 S L32 AND (BRAIN)
L36             0 SEA FILE=MFE SPE=ON  ABB=ON  PLU=ON  L35 AND PD<2002
                DIS IBIB ABS L6 1-5
                DIS IBIB ABS L9 1-3
                DIS IBIB ABS L23 1-5
                DIS IBIB ABS L26
                DIS IBIB ABS L28
                DIS IBIB ABS L31
COST IN U.S. DOLLARS                            SINCE FILE      TOTAL
                                                  ENTRY    SESSION
FULL ESTIMATED COST                               153.64     153.86

STN INTERNATIONAL LOGOFF AT 12:13:52 ON 17 DEC 2009
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